has not the hereditary functional ability to meet at least a considerable part of her infant's growth and development needs should not have her life made miserable in an attempt to accomplish the impossible. On the other hand, breast feeding should not be discarded too lightly without a fair attempt to bring the breasts up to their functional capacity. Virgin breasts must be stimulated sufficiently often by thorough emptying to establish full function. Double breast feeding (the value of which was understood and made use of by our grandmothers) is a very valuable means to this end. The trial period must be extended over a sufficient time to prove the absence of the ability to carry on nursing satisfactorily. Six weeks postpartum is the shortest period which can be accepted as sufficient for demonstrating the lack of an amount of breast milk that is valuable as entire or partial nourishment for the infant. Many mothers can discard supplemental feedings at the end of this time and continue over a long period of very satisfactory milk production.

The intangible values of breast feeding over artificial feedings in the after-life of the infant are not easily made apparent and may not be important. Man's life is long and is influenced by many factors and conditions. Nevertheless, they may well be considerable. The psychic satisfaction that comes to the mother who nurses her infant is real and apparent and no doubt serves to increase and enrich her attachment to her child.

The desire to nurse her infant is well worth all the encouragement and painstaking help which her physician can give to the mother.

RECURRENT RETINAL HEMORRHAGES*

REPORT OF CASES

By THEODORE C. LYSTER, M. D.
Los Angeles

DISCUSSION *by M. F. Weymann, M. D., Los Angeles; Joseph L. McCool, M. D., San Francisco; Hans Barkan, M. D., San Francisco.*

RECURRENT retinal hemorrhages, especially those occurring in young adults, although not frequently reported in ophthalmic literature, are believed not so rare in practice. Trauma, lues, or a probable focal cause, other than pulmonary, can reasonably be excluded in a great many cases, leaving a relatively large group with undetermined etiology.

TUBERCULOSIS AS A CAUSATIVE FACTOR

Many of these may be due to a chronic tuberculous retinitis. These are the tragic cases for the oculist. One eye is usually lost, or permanently damaged, and the second eye on its way to becoming blind before the low-grade changes, either in the tracheobronchial glands, or other pulmonary structures, generally at the hilus, are considered as possibly responsible for the eye condition. Because the physical signs in the chest are usually not marked, these patients are rarely seen in sanitaria for the tuberculous. The internists in general, and especially those interested in tuberculosis, are exceedingly skeptical about the pulmonary changes having any direct association with the ocular disturbance. Our attention has been repeatedly called to this disassociation, especially by Jackson [1] and Finnoff.[2] That this is so hardly seems reasonable in view of our present knowledge of tuberculous conditions. It would hardly appear necessary to anyone following the trend of thought, as seen in the mass of literature on tuberculosis, to doubt that a latent pulmonary infection, such as is frequently seen in a peribronchial lymph node, may be the cause of the presence of a lesion in a far-distant organ of the body. The papers of Ophüls[3] and Krause[4] might here be mentioned simply to support this statement. Opie[5] has well stated: "Anatomic evidence furnishes abundant proof that the tuberculosis of the healthy should not be regarded as a trivial infection, of interest only to the pathologist."

Nearly every tissue of the body would appear susceptible to a secondary tuberculous manifestation, even when the primary lesion is almost negligible. Every structure in the eye—even the lens—has been found tuberculous at times. As for retinal tuberculosis, it has been medically accepted for many years and recognized as a vascular lesion, generally associated with superficial or deep retinal hemorrhages. Except when secondary to an extension from a neighboring structure, usually the choroid, the primary focus has frequently been suspected rather than determined. Friedenwald,[6] in discussing recurrent retinal hemorrhages, concluded that "none of these lesions are specifically tuberculous," which seems to be the generally accepted opinion, from its pathology. However, again quoting: "The pathology of phlyctenular disease shows little that is specific." Both conditions, clinically, are frequently considered tuberculous, but there seems to exist, even among pathologists, a willingness to accept the latter as an allergy but not the former. It would appear from our unsettled knowledge of allergy and immunology that no positive conclusion is warranted at the present time. Because of the comparative rarity where the tubercle bacillus has been found in suspected chronic retinal tuberculosis, much controversy has resulted to explain this rarity. Judging from the work of Otori [7] in 1914, and confirmed by many since (especially Finnoff [2]), it is exceedingly difficult to produce a primary tuberculous retinitis in animals, even by injection of tubercle bacilli into the carotid artery or temporal vein. The influence created by the classic work of Rosanow [8] that an organism such as the *Streptococcus viridens* from an apical abscess must be present in an ocular tissue before a focal reaction for the eye can be produced, still dominates our present general concept of all allergic ocular reactions.

Because of accessibility much animal experimental work has been possible in phlyctenulosis. The absence here of tubercle bacilli in the sclerocorneal tissue would appear confirmed, and this shakes somewhat our assurance that even in tuberculous retinitis, where the organism is so rarely found, that the living organism is a necessity. While much of what has been stated might appear simply controversial, the underlying thought is that proven pathology and clinical experience seem to differ at the expense of the

* Read before the Eye, Ear, Nose, and Throat Section of the California Medical Association at the fifty-ninth annual session at Del Monte, April 28 to May 1, 1930.

patient. This is especially so if the lesion is truly one of tuberculous origin, for irreparable damage closely follows the appearance of these ocular disturbances. Whatever may be the verified results as to the mode of production of chronic tuberculous retinitis—whether an allergic reaction with or without the actual presence of the tubercle bacilli in the retinal tissues—the pathologic changes are quite definite. Finnoff [2] states as follows in describing retinal tuberculosis:

"The condition occurs as a vasculitis or a perivasculitis of the retinal vessels, or as small white areas in the retina which resemble the exudates found in albuminuric retinitis. The vessels, when involved, frequently rupture, and hemorrhages into the retina and vitreous occur. Hemorrhages absorb slowly and are frequently replaced by fine strands of scar tissue (retinitis proliferans). The hemorrhages are usually recurrent. Following hemorrhages the vision is markedly impaired. The amount of impairment depends upon the extent and location of the bleeding. Both eyes are usually involved in this process, and the prognosis regarding vision is poor. This type of tuberculosis as a rule is of the chronic variety, and it is frequently seen in persons with latent tuberculosis. It is often impossible to find the primary seat of the disease, as these patients appear to be perfectly healthy.

"The picture is essentially one of vascular pathology and differs both in character and development from that seen in the acute forms of intra-ocular tuberculosis.

"In the acute type of intra-ocular tuberculosis, active tuberculosis is usually found elsewhere in the body and the ocular condition is due to the presence of living tubercle bacilli. If the patient lives for a long enough period after the eye condition develops, a destructive ocular process will be observed. When the lesions are inside the eye, the involvement rapidly progresses until the eyeball is entirely destroyed, or until it ruptures and its contents are expelled. While no difficulty is encountered in locating tubercle bacilli in the acute cases, only with the greatest rarity have the actual tubercle bacilli been found in the chronic types."

Otori [7] reported two cases where sections were made, showing stained tubercle bacilli in the sheaths of retinal veins. Fuchs [9] reports one case of tuberculous disease of the sheaths of the retinal vessels found at autopsy; the bacillus evidently was not found as it was not mentioned. Holloway [10] describes in detail the course of two cases of definite tuberculous retinal phlebitis associated with recurrent ocular hemorrhages. No mention, however, was made of his finding the actual organism present in the lesions. As a result of experimental work done in 1914 by Otori [7] his conclusions were:

"The rarity of primary retinal tuberculosis depends not only on the small blood volume or the greater velocity of the blood stream, but rather is to be ascribed to a certain indispositional attitude of the retina toward a tuberculous primary affection.

"The experiments show further perivascular findings, not only in the choroid but also in the other organs of the body, especially the lungs.

"The primary process in the cases before us began as a perivasculitis of the retinal veins, and this perivasculitis is not a symptom of reaction against the toxic irritation, but is caused directly by the tubercle bacilli which reached the retina by way of the lymph.

"In my judgment, the investigations as to the existence of tubercle bacilli in the retina offer no such great difficulty as other authors assume."

The intervening fifteen years in ophthalmic literature would not appear to confirm such ease in finding this organism, as he seems to be the only one so fortunate.

Verhoeff [11] in reporting his histologic findings in a case of localized tuberculous chorioretinitis, while failing to find tubercle bacilli in stained sections, based his diagnosis of tuberculous chorioretinitis on the nodular arrangement of epitheloid and giant cells opposite practically every retinal vein, but nodules also occurred without apparent relation to the veins.

Such evidence, however limited, is conclusive as to the fact that tubercle bacilli at times do lodge near retinal veins and are associated in such cases with recurrent retinal hemorrhages. Whether the not infrequent cases of recurrent hemorrhages in youth are always due to the actual presence of the organism cannot be accepted without more confirmatory data than now seems available.

Weekers,[12] in a recent experimental treatise on phlyctenulosis of the eye and tuberculosis, came definitely to the conclusion that these conditions are usually tuberculous in origin and focal in character; are allergic manifestations, the result of a latent pulmonary tuberculosis. If this is true it would appear reasonable that the general allergic principles may be the same for a retinal focal reaction, as for a corneal one.

Because the eye of man in an early active stage of recurrent retinal hemorrhage of probable tuberculous origin so rarely is enucleated and studied in serial section, the proof of its tuberculous nature is most difficult to establish. Even when such eyes are obtained, and when sectioned, and no giant cells, caseation or tubercle bacilli are found, there still remains a possibility that the cause may still be a tuberculous focal reaction. That focal reactions may occur in an apparently normal eye in a patient with pulmonary tuberculosis has been well recognized for many years. Calmette's conjunctival reaction following topical application of tuberculin was formerly quite generally used and then condemned, not because of not giving information, although probably of little diagnostic value, but because it resulted at times in the unnecessary loss of an eye. With the wide use of tuberculin there is now recognized a constitutional, a local, and a focal reaction (hemorrhage and exudate), whether the location be the eye or any other part of the body.

Krause [5] states: "All tissues of a tuberculous animal are allergic (Nichols on the lung, Kimberg on the kidney, Peterson on the pleura, Soper on the liver, etc.)." Again: "While the comparative allergic capacity of uninvolved tissue is a problem awaiting solution, there is no question of the much greater reacting quality of tuberculous foci as compared with nontuberculous tissue of the same animal."

ACTION OF TUBERCULIN

Everyone who has used tuberculin in these retinal conditions will admit that the ever-present danger from too large doses is a focal reaction.

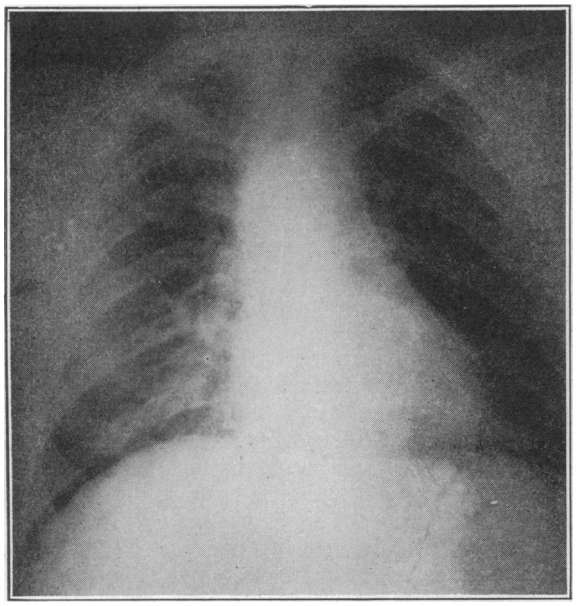

Fig. 1 (Case 1).—Radiograph of the chest shows normal adult lung fields with moderate increase in the thickness and density of the hila and peribronchial shadows, especially in the right lower lobe.

While it must be admitted that a metastatic tubercle of the retina would probably be activated by an overdose of tuberculin, it is also probable that a retinal tubercle thus activated would not, as a general rule, so readily and completely subside, leaving often hardly a trace of its former presence. This is so frequently seen following repeated focal reactions in the retina from tuberculin. Because of the not infrequent serious damage to a susceptible eye, the dose of tuberculin, according to Finnoff,[2] "should be controlled by the focal reaction and not by the local or constitutional reaction—the latter, such as Pirquet, Moro, and Calmette tests, being valueless as diagnostic tests in eye tuberculosis, showing only that the individual is sensitive to tuberculin or is recovering from an old tuberculous infection."

Certain pathologic phenomena are common in practically all of these patients with recurrent retinal hemorrhages. There are present low-grade hilar changes, suspicious of tuberculous origin, demonstrated roentgenologically even when not by auscultation or percussion; they are more frequent in the second and third decades of life and, statistically at least, more among men than women. A focal reaction can only too frequently be produced by an overdose of tuberculin, or even by a disturbance such as an acute cold, which acts as a pulmonary excitant. There are frequent exacerbations, usually with some apparent pulmonary disturbance, however slight. Recovery may be practically complete temporarily, or go on to absolute blindness and ocular degenerative changes—rarely, however, with caseation or other evidence of manifest tuberculosis of the eye. The lesions are vascular, affecting the retinal veins and not the retinal arteries.

The retinal lesions, especially in the deeper layers, are usually in both eyes and quite similar as to location and character, although one eye is generally more advanced than the other. This phenomenon, while quite the rule with toxic disturbances, could only occur as a coincidence by metastasis.

PATHOLOGIC CHANGES IN THE RETINA

The pathologic process as seen by us ophthalmoscopically, begins with some dilation and thickening of the coats of one or more veins; then an oozing extravasation takes place at one or more points. The bleeding in the retina may be superficial or deep. When superficial, it may

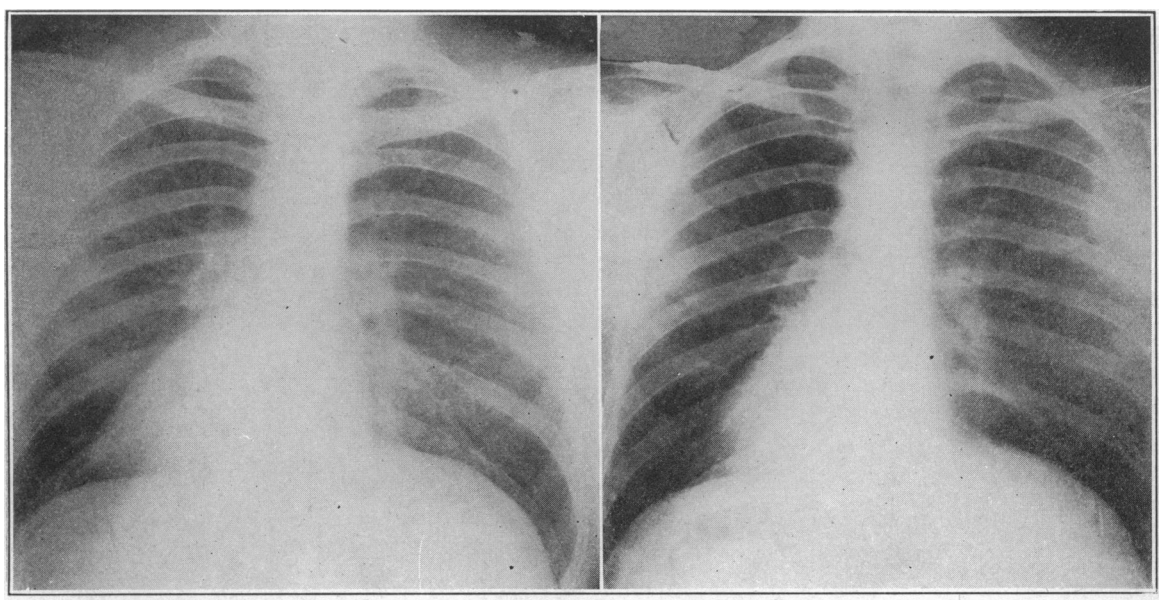

Figure 2a, Case 2. Figure 2b, Case 2.

Fig. 2a (Case 2) and Fig. 2b (Case 2).—(Illustrations here are reversed, showing the heart on the right side. The plates should be turned when the prints are made.) Radiographs of the chest show moderate generalized thickening, with peribronchial and hila shadows on both sides. Aside from this the appearance is that of a normal chest.

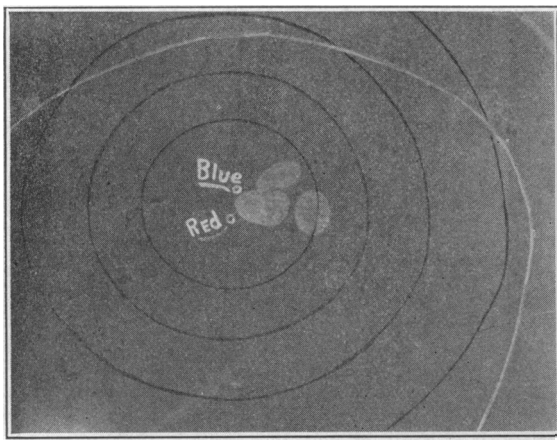

Fig. 3a (Case 3).—Absolute scotoma in the left half of the field of the left eye, which appears as an irregular enlargement of the blind spot. In other respects the field is normal. (Please note that here in the woodcuts 3a should be 3b, and 3b should be 3a—in other words, they have misplaced the visual field photographs.)

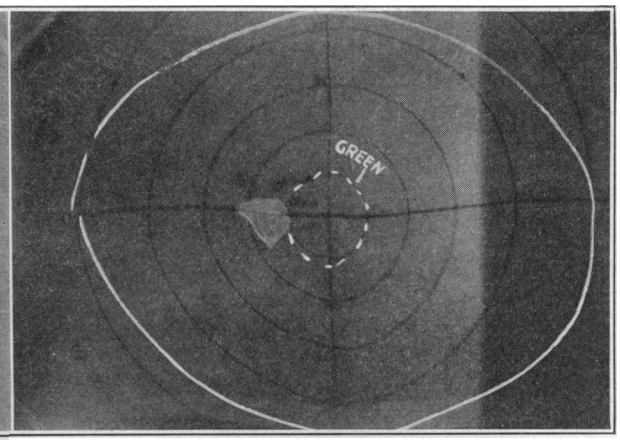

Fig. 3b (Case 3).—Three well-marked absolute scotomata in the right field of the right eye. The larger appears to be an extension enlargement of the blind spot; the other two, one above and one below, are inside the twenty-degree circle. In other respects the fields are normal.

break into the vitreous. Later this exudate may become organized, persisting as floating opacities in the vitreous. They are frequently attached by long fibrous bands to a retinal vein, which latter may be more or less damaged. The vein is usually not obliterated, but its course and caliber much altered. Frequently yellow-white bands extend outward from the disk along the course of the retinal vessels as evidence of retinal folds and fibrous retinal changes (retinitis proliferans). When the hemorrhage and exudate extends toward the choroid the resultant lesion is frequently an atrophy of deep retinal and choroidal structures, leaving only sclera covered by superficial retinal layers. These areas are often surrounded by pigment and not infrequently a retinal vessel is seen to course over the white patch uninterrupted in size or direction. These lesions are often located at corresponding points of each retina. If one is macular, both are; if one adjoins the disk margin, the other does likewise. The end result is one of atrophy following a limited retinochoroiditis, or scar tissue along the course of the affected vessels with the visual damage depending upon the location of the lesion and its extent.

RÉSUMÉ

Recurrent retinal hemorrhages in the young adult are sufficiently frequent and tragic to require early diagnosis.

They are generally due to a phlebitis. One eye is frequently lost or severely damaged, and the other eye on its way to permanent injury, or even blindness, before its probable tuberculous nature is actively suspected.

Eyes with these retinal lesions, in early active stages, are rarely enucleated and then studied in serial sections.

While they are at times due to a metastatic ocular infection, wherein tubercle bacilli have been found in stained sections following enucleation, there is much clinical evidence to support the contention that these lesions may often be a tuberculous allergic manifestation of pulmonary origin. The mode of production, however, is of less importance than its early recognition if useful vision is to be retained.

Regardless of the apparent trivial character of the pulmonary lesions, these patients should receive early and active specific treatment, such as is now common in our modern sanitaria for the tuberculous.

The closest coöperation between the internist and oculist is essential, both for diagnosis and treatment.

Tuberculin is of recognized value, but must be used with great care and a focal reaction avoided because of the danger of serious permanent visual damage.

While admitting the appearance of the pulmonary lesions may show little that is not seen in the average healthy individual, the added presence of recurrent retinal hemorrhages before middle life, when trauma, lues, and focal infections other than pulmonary can be reasonably excluded, a tuberculous origin should be suspected and given the benefit of a doubt by active specific therapy.

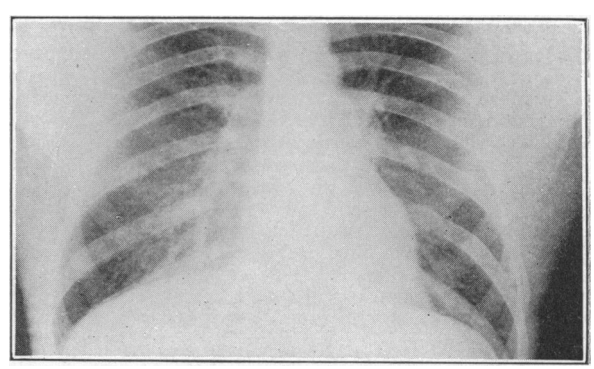

Fig. 3 (Case 3).—Radiograph of the chest shows practically normal adult lung with moderate increased density of hila and peribronchial glands shadows.

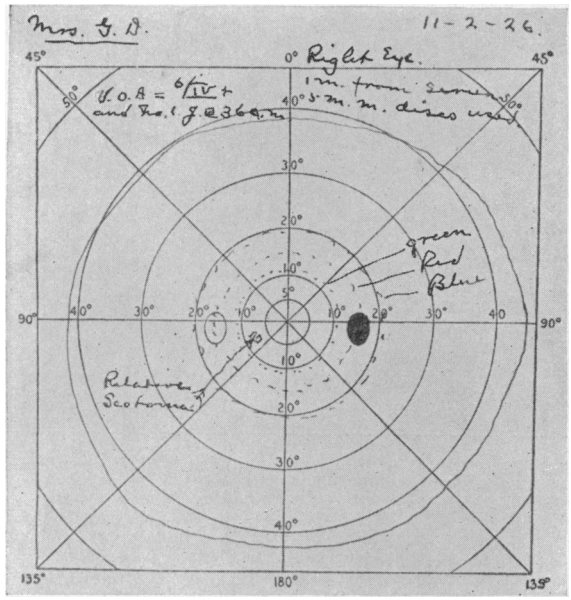

Fig. 4 (Case 4).—Visual field of right eye normal in all respects with the exception of a small relative scotoma in the inferior nasal quadrant at about seven degrees from the point of fixation.

Four case histories are submitted to illustrate the two common forms of probable chronic tuberculous retinitis associated with recurrent retinal hemorrhages.

REPORT OF CASES

Case 1.—W. B. Man. Born in 1900.

Family History.—Negative.

Personal History.—Usual childhood diseases; tonsils out; x-ray of sinuses clear; teeth normal. Laboratory findings as to blood and spinal Wassermann, renal function, basal metabolism, blood chemistry, urethral smears, showed no marked pathology. Positive Von Pirquet. Constipated; nose-bleeds; general physical findings—those seen in an active normal young man with the exception of low-grade pulmonary changes with areas of fibrosis in both lungs.

Ocular History.—On September 1923 had recurrent retinal hemorrhages in left eye. Eye was finally enucleated two years later because of inflammatory changes. Report on sections made by Doctor Finnoff shows changes suspicious of tuberculous origin, but not sufficiently characteristic for a positive diagnosis. No tubercle bacilli found.

In February 1927, multiple retinal hemorrhages occurred in right eye, largely confined to the superior nasal vein. Vision reduced to light and shadow only, for a period of nine months, during which time he had learned the use of Braille.

In October 1927, following gradually increased doses of B. E. tuberculin, using the method in practice at the Knapp Memorial Hospital, vision gradually increased with the clearing of the vitreous and cessation of hemorrhages. Vision now 6/XV and No. 1 Jaeger at 20 centimeters, with his original lens correction. Fundus picture that of retinitis proliferans with organized exudate in the vitreous; an elongated and distorted disk and long narrow bands of apparently scar tissue extending radially upward along the tracts of former blood vessels. Periodic recurrent hemorrhages have taken place, usually associated with an acute cold or overexertion. Vision on February 1930: 6/IX and No. 1 Jaeger at 30 centimeters. Vitreous clearing from a recent hemorrhage, following a strenuous trip to a high altitude. Prior to last hemorrhage the vitreous was clear with the exception of a few small floating patches of organized exudate which had become attached to one of the tributaries of the superior nasal vein. This vein for several disk diameters, prior to its union with the superior temporal vein, is bordered by bands of scar tissue in places, distorting but not markedly obstructing the venous flow. The remainder of the retina and disk appeared normal. At no time have the retinal arteries been disturbed to any appreciable degree. For roentgenograph of chest, see Fig 1.

Case 2.—G. V. Man. Born in 1900.

Family History.—Negative for tuberculosis, except mother's sister died of a pulmonary disturbance at the age of thirty-five. A sister and a brother died in infancy of "brain fever."

Personal History.—Usual childhood diseases. Tonsils out. Occasional nose-bleeds.

Ocular History.—No eye trouble until April 1920, when he noticed lines and crosses disturbing vision of left eye. No pain, but slight headache. Vision decreased rapidly to light and shadow only in this eye and suggested seeing through a red cloud. One month later the vision of the right eye became affected; a ring-shaped blur, red in color, was noticed by him floating before the vision of this eye. Vision now light and shadow only. A thorough physical and complete laboratory examination was essentially negative after admission to the Barlow Sanitarium in January 1926, except suspicious areas of fibrosis of each lung. Under observation he was found to run an evening temperature elevation and finally tubercle bacilli were found in his sputum. Von Pirquet positive.

Ocular Findings in September 1927.—Right eye: Anterior segment normal. Right fundus: Vitreous clear except for three small, and one large, floating masses of organized exudate, each attached to a retinal vein by a long fine fibrous band. There was present a small retinal area in the superior nasal zone suggestive of possible detachment, but not progressive. Both the superior nasal and temporal veins peripherally were markedly disturbed in their course. These veins, for a disk diameter, were shrunk to one-third their normal size, but still carried blood and were apparently normal on each side of the lesion. The retina in the immediate neighborhood was considerably altered in structure, being granular in appearance and containing many fine white lines, evidently scar tissue. Along the course of the inferior ophthalmic vein were small areas of retinochoroidal atrophy—some pigmented, some nonpigmented—but all quiescent and about the diameter of the vein in size. Vision: 6/VI plus and No. 1 Jaeger at 36 centimeters. Vision in left eye improved; now 4/XXX. Anterior segment normal except iris had changed color to a greenish from a blue-gray, and small vacuoles were seen deep in the lens structure. A mass of floating exudate in the vitreous, otherwise clear, prevented details of the central and superior nasal areas being clearly seen. The disk appeared elongated vertically and a band of organized exudate extended downward for three disk diameters along the inferior vein, whose walls were thickened and at times covered. Peripherally, these veins appeared normal. This young man has had no definite hemorrhages in the last year while employed regularly at gas-filling stations. Tuberculin B. E. has been continued for a period of over four years with apparently only favorable results. For roentgenographs of chest, see Figs. 2a and 2b.

Case 3.—H. A. J. Man. Age, 32.

Family History.—Negative for tuberculosis.

Personal History.—Usual childhood diseases. Tonsil and adenoid operation in childhood. Second tonsillectomy in 1926. When in college right antrum was washed out and abscessed tooth removed. Put on glasses in 1925 for relief of headaches. Has been subject to frequent acute colds all his life. In January

1924, acquired an acute cold, which persisted and extended to left ear and left ethmoid, requiring drainage. Noticed a black spot before vision of right eye when reading. A little later was found to have a retinal hemorrhage into the vitreous of this eye. This was considered of tuberculous origin and he was placed on gradually ascending doses of tuberculin. Was carefully studied by well known oculists and internists. Vision improved to 6/IX and there was noted an area of acute retinochoroiditis, temporal to the disk and of a similar size, which later subsided to an atrophic area with a little pigment along its margin and several radiating lines of atrophy. Visual field showed paracentral scotoma (Fig. 3). Following an overdose of tuberculin, a second hemorrhage resulted, but there has been none since in this eye. Von Pirquet positive.

Two years later noticed a black spot, disturbing the vision of the left eye, which was found to be due to some eight venous punctate hemorrhages in an area temporal to the left disk. A true counterpart as to location of the lesion of the right eye. The hemorrhages rapidly absorbed, leaving only fine granular changes in this area. A slight enlargement of the blind spot was up and in, which soon disappeared. Vision again normal for him. No atrophic changes visible. Disturbance apparently confined to deep retinal structures.

Three years now have elapsed under tuberculin therapy, with no recurrence. Several physical and complete laboratory findings have proved negative except for the upper respiratory disturbances already noted; Von Pirquet plus, and low-grade changes at the base of each lung, probably tuberculous, though apparently quiescent when last examined. For roentgenograph of chest and visual fields, see Figs. 3a, 3b, and 3c.

* * *

CASE 4.—Mrs. G. D. Age, 33.

Family History.—Negative for tuberculosis.

Personal History.—Usual childhood diseases. Frequent colds all her life. Tonsils out. X-ray of teeth negative. In 1920 general physical and extensive laboratory examinations negative, except a tentative diagnosis of incipient tuberculosis.

Ocular History.—Two years later, synchronizing with the birth of her second child, suddenly lost the central vision of her left eye. Could count fingers at two feet, seen eccentrically. Sight has never improved. Three years later (1925), following a severe cold, noticed the nasal field of her right eye was blurred. Cleared gradually but a year later recurred, this time affecting her central vision. On looking at the eyes of a person's face, the nose appeared blurred.

Ocular Examination.—On November 18, 1926: Vision O. D. 6/VI plus and No. 1 Jaeger at 36 centimeters, with distortion of vertical lines. O. S.—Fingers, eccentric, at two feet. Anterior segment in each eye normal. Vitreous clear. Right fundus normal except macular region. The fovea is blurred by a granular appearance of the retina, suggestive of low-grade inflammatory condition now quiescent. Left fundus showed a white, punched-out area in the macular region, about one-half disk in diameter and surrounded by pigment, evidently choroidal atrophy. Left eye remained unchanged, but right eye improved to 6/IV and practically disappearance of distortion. For visual field of right eye, see Fig. 4. Physical and laboratory findings otherwise negative.

1930 Wilshire Boulevard.

REFERENCES

1. Jackson, Edward: Intraocular Tuberculosis, Ophthalmic Record, 25:1 (Jan.), 1916; Present Understanding of Tuberculosis, J. Indiana M. A., 17:403 (Dec.), 1924.

2. Finnoff, William C.: Tuberculosis of the Eye, Am. Rev. Tuberc., 7:67-80 (April), 1923; Recurrent Hemorrhage into the Retina, J. A. M. A., 79:939 (Sept. 16), 1922; Differential Diagnosis in Tuberculosis of the Eye, J. A. M. A., 90:341 (Feb. 4), 1928; Changes Found in Eyes of Rabbits—Injection of Living Tubercle Bacilli, Am. J. Ophth., 7:8189 (Feb.), 1924.

3. Ophüls, William: A Statistical Survev of Three Thousand Autopsies, Stanford Univ. Pub. Med. Sciences, 1, No. 3, 173, 1926.

4. Krause, Allen K.: Significance of Allergy in Tuberculosis, Tr. Nat. A. Prev. Tuberc., 17:348, 1921.

5. Opie, Eugene L.: Tuberculosis of the Lungs and Bronchial Lymph Nodes., J. A. M. A., 89:1293 (Oct. 15), 1927.

6. Friedenwald, J. S.: Pathology of the Eye, p. 80. The Macmillan Company. 1929.

7. Otori, Kaizo: Pathologish Anatomisch—Netshautuberkulose, Arch. f. Augenh., 79:22, 1914.

8. Rosanow, E. C.: Elective Localization of Streptococci, J. A. M. A., 65:1687 (Nov. 13), 1915.

9. Fuchs, Ernest: Textbook of Ophthalmology, eighth edition, 651. Lippincott Company. 1924.

10. Holloway, T. B.: Relationship of Tuberculosis to Certain Fundus Changes, J. Iowa Med. Soc., 15:629-637 (Dec.), 1925.

11. Verhoeff, F. H.: Histologic Observations in Case of Localized Tuberculous Chorioretinitis, Arch. Ophth., 1:63-70 (Jan.), 1929.

12. Weekers, L.: Phlyctenes Ocularies et Tuberculose, Arch. D'ophth., 44:342 (June), 411 (July), 1927.

DISCUSSION

M. F. WEYMANN, M. D. (2007 Wilshire Boulevard, Los Angeles).—The mechanism producing phlyctenules can hardly be considered identical with that causing recurrent hemorrhages into the vitreous, as the two conditions do not parallel each other. Weeker's statistics definitely show that the peak of the curve for incidence of phlyctenules occurs in early childhood, while there are slight rises at the age of puberty, and during the menopause. This condition also greatly predominates in the female sex. On the contrary, recurrent hemorrhages occur most often in males and usually between the ages of eighteen and thirty. Marked chronic constipation is so often an accompanying factor of recurrent hemorrhages that it must also bear a rather important etiological relationship. By again calling our attention to the consideration of tuberculosis in these conditions Doctor Lyster has done us a valuable favor.

*

JOSEPH L. MCCOOL, M. D. (1319 Four-Fifty Sutter, San Francisco).—Recurring vitreous hemorrhages as a distinct disease was first described by Von Greafe. Later Eales made some observations on the disease and it is often referred to as Eales' disease.

The etiology and symptomatology have been fully covered by the essayist.

With regard to the former, however, it is not always easy to classify these cases. While it is probably true that in many cases the vasculitis and perivasculitis of the retinal vessels are tuberculous, nevertheless I do not believe that all cases may be so classed.

Eales considered that intestinal toxemias played an important rôle. Zentmeyer and others believe that endocrine disturbances are responsible for some cases.

I can recall two cases which I believe illustrate this divergence in etiology. Parenthetically it might be said that, while it is sometimes very difficult to say that a certain case is due to tuberculosis, it is also wrong to assume that our inability to find a primary focus rules out that disease.

Some years ago a surgeon brought his son, aged fourteen years, for an examination of his one remaining eye. From the history I gathered that the boy had had a severe intra-ocular inflammation which necessitated the removal of one of his eyes. The re-

maining eye was the seat of recurring vitreous hemorrhages which at the time of the examination had reduced his vision to approximately 10/200. Lues as a cause had been eliminated, all known sources of focal infection had been removed but no tuberculin test had been made. He responded positively to this test and was promptly put on small doses of B. E. gradually increased.

His vision increased to 20/30. He was sent back to school and when last heard from had had no recurrences.

The other case was a woman, aet. thirty-two years. Vision in the right eye, 5/200; left, 20/25. This woman had recurring vitreous hemorrhages in the right eye. She had been given a course of tuberculin, but with no benefit. There were several rather interesting features which developed from the history:

1. Patient had a severe fright during a menstrual period which ceased. Almost immediately afterward, the first vitreous hemorrhage occurred.

2. Every hemorrhage which the patient has ever had came at the menstrual period.

3. If the period is delayed two days or more, the patient has a fresh hemorrhage.

Within three years, four times the vision was better for a time; three of these times occurred during a pregnancy.

The etiology in this case was probably endocrine.

❦

HANS BARKAN, M. D. (490 Post Street, San Francisco).—In private practice juvenile retinal and vitreous hemorrhages have been rare cases for me. I have seen more patients in the late thirties and early forties with retinal hemorrhages not explainable on any of the usual grounds. It has also not been my experience that the male sex predominates; a good number of such patients have been women. These facts only show that one man's material may shape itself quite at variance for some reason from the accepted standards, and conclusions derived from this experience only should not be hastily drawn.

In typical cases there is little doubt but that a slow tuberculous focus located in the various walls is the etiological factor. The clinical observations of Stock and Axenfeldt, Miller and Finnoff, and the pathological studies of Fleischer and Ranke all point to this. In severe cases everything may be over before we can aid. I have seen such hemorrhages massive and recurring every few days for weeks. In recurring smaller retinal hemorrhages tuberculin is worth while. Local reactions must absolutely be avoided.

Doctor Lyster has well covered an important field. In his hands and in other competent ones such cases are usually well treated. The wholesale merchant in ophthalmology either misses them, or, still worse, makes this diagnosis in all ocular conditions of uncertain etiology to the fright of his patients and the depletion of their purse.

❦

DOCTOR LYSTER (Closing).—Doctor Weymann has pointed out differences as to incidence in phlyctenulosis and retinal hemorrhage. There is, however, a suggestive similarity in etiology.

Although chronic constipation is a frequent accompanying condition, it is by no means a constant factor and probably influences the ocular disturbance as would some focal infection.

I wish also to thank Dr. McCool and Dr. Hans Barkan for their discussions. It is fully realized that neither the etiology nor its manner of producing the retinal disturbance has been definitely determined. Clinically, however, based on some known pathology and ophthalmoscopic observations, there is considerable evidence of a probable close relationship between recurrent retinal hemorrhages of the adult and tuberculous infections. The final decision will only be made when definite pathologic proof is available.

THE LURE OF MEDICAL HISTORY

PALMARIUS (PIERRE PAULMIER)

By FELIX CUNHA, M. D.
San Francisco

IT is interesting to note that as far back as three hundred years ago anyone who dared propose any radical change in the accepted methods of treatment of the day was meted out punishment that was not only swift, but severe.

Palmarius (Pierre Paulmier), born in 1568, the nephew of an illustrious uncle-physician, who had served with Paré in the French campaigns, studied in Paris and received his license in 1596. In 1608 he published his famous "Lapis Philosophicus Dogmaticorum," the frontispiece of which is reproduced here. This work so enraged the Galenists of the time that a censure was passed upon it and its author by the medical faculty of Paris on the 28th of January, 1609. The author had taken sides with the adherents of the theories of Paracelsus and advanced the merits of the treatment of disease with preparations of gold and antimony, but particularly advocated the use of antimony. In its citation the Faculté de Paris condemned the book as being "full of errors, deceits, impostures, and lies, and

LAPIS
PHILOSOPHICVS
DOGMATICORVM.

Quo paracelſiſta Libauius reſtituitur, Scholæ Medicæ Pariſienſis iudicium de Chymicis declaratur, Cenſura in adulteria & fraudes Parachymicorum deffenditur, aſſerto veræ Alchemiæ honore.

Per P. Palmarium Doctorem Pariſienſem Galeno-chymicum.

Ad Illuſtriſſimum Cardinalem Perronium.

Adiecta eſt Hiſtoria Læproſæ Mulieris Perſanatæ.

PARISIIS.
Apud DAVIDEM DOVLCEVR, via Iacobea ad Mercurium inuolucrem.
1624.
CVM PRIVILEGIO REGIS.

Fig. 1.—Title page.